VET
RECEIVED
JAN 15
VET. MED. LIBRARY
JAN 12 1959

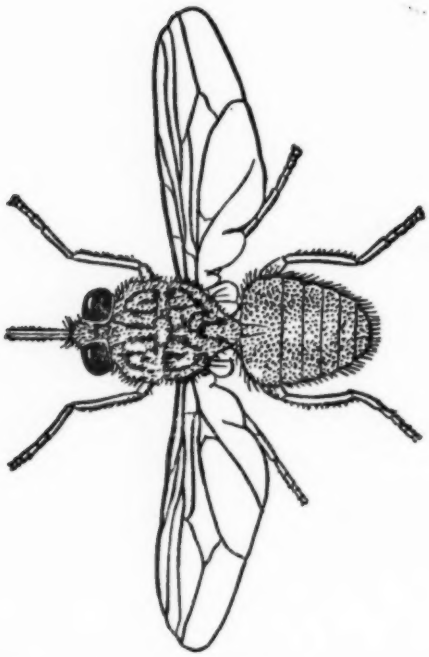
THE JOURNAL OF THE BRITISH VETERINARY ASSOCIATION

The Veterinary Record

PATRON: HER MAJESTY THE QUEEN

Vol. 70 No. 51 Issue No. 4211 1203-1218

December 20th, 1958



PROTHIDIUM

the soluble drug
for the prophylaxis
of bovine trypanosomiasis

Prothidium is available as 0.5G tablets in bottles of 20 and 100.

Further information gladly supplied on request.

Boots **PROTHIDIUM**

—another product of Boots research

BOOTS PURE DRUG COMPANY LTD.,
International Division, NOTTINGHAM, ENGLAND

The NEW S.E.T.A. Mk. III Veterinary Model has shown itself an effective treatment for strain, sprain, contusion and rheumatism by rhythmic muscular contractions (faradism).

Does NOT generate heat in any form

Can be operated with a wall-attachment from a separate 12-volts accumulator, or, carried in the luggage space of a motor car, on a 12-volts car battery.

Full particulars:—

S.E.T.A. LIMITED

Critchmere Lane, Haslemere, Surrey, England.

The
VETERINARY RECORD

Founded by William Hunting, F.R.C.V.S., in 1888

7, MANSFIELD STREET, PORTLAND PLACE, LONDON, W.1. MUSEUM 6541

No. 51 DECEMBER 20th, 1958 Vol. 70

Registered for Transmission as a Newspaper *Price 2s. post free*

"Chorea" of the Dog and Lignocaine

BY

J. F. WHITTLESTONE
Sheffield

SUMMARY.—Nineteen chorea subjects were given lignocaine, 18 subcutaneously and one intramuscularly. Thirteen showed complete relief of symptoms. Opportunity arose in 2 of the 6 showing no response to repeat the dose intrathoracically when complete relief of symptoms was seen. A third resistant subject had an increased dose given subcutaneously which produced double rate and half amplitude of the twitch, and also vomiting and further inco-ordination. Absorption of 10 per cent. lignocaine from the subcutis seems to be occasionally too slow.

Of 4 normal dogs given 1 ml. of 10 per cent. lignocaine per 10 lb. bodyweight subcutaneously, 1 showed slight inco-ordination and vomited.

Introduction

THIS work follows up a chance observation that a local anaesthetic, lignocaine, suppressed the twitches of "canine chorea."

Whittier (1956) and McGovern *et al.* (1948) summarised existing knowledge of "chorea" of the dog and suggested "Flexor Spasm Syndrome" and "Canine Tremor-rigidity Syndrome" to replace a proved misnomer. Both suggestions seem so inept that the established if erroneous "chorea" is used here.

Classification

A classification of canine choreas was necessary to give a brief and accurate description. This was attempted on anatomical grounds with lists of twitching muscles, but the scheme was found to be hopelessly complex. The classification developed used terms which are defined below:—

"Settled" case. No change in type or distribution of twitches for at least 6 weeks.

"Not settled" case. Twitches obviously spreading or not established without change for arbitrarily fixed 6-week-period.

Type "A." Twitches apparent when dog is at rest only.

Type "B." Twitches apparent when dog is at rest and standing but not when walking.

Type "C." Twitches present all the time.

A commonly occurring description is "Settled left foreleg Type C." These cases have shown identical anatomical distribution of twitches—outstanding among them being a disablingly powerful twitch of the adductor muscles. Similar comparisons show that this kind of description gives a picture complete enough for results to be compared.

Methods

A series of observations quickly showed the basic facts of lignocaine action: ½ ml. 3 per cent. lignocaine per 10 lb. bodyweight, intravenously, eliminated twitches within 2 minutes, but all twitches returned in 20 minutes to 1 hour after dosing. 1 ml. 2 per cent. procaine per 10 lb. bodyweight, intravenously, removed twitches which returned in 20 minutes to 1 hour, but induced some hypnosis.

Given by mouth as a draught, an active amount was absorbed from the buccal mucous membrane; similar amounts given in gelatine capsules showed no activity.

Significant amounts of lignocaine given subcutaneously to a dog with A, B, and C type twitches showed that A twitches disappeared quickly; B and C types appeared to double their frequency with reduction of amplitude before disappearing. As the effect of lignocaine faded this chain of events was repeated in reverse.

Lignocaine does not act on motor nerve endings in muscle. A dog showing B type in all 4 legs, was given lignocaine intravenously with a tight rubber ligature above the elbow of a foreleg. The twitches faded at the same rate on all 4 legs.

For rapid collection of information a standard dosage of 1 ml. of 10 per cent. lignocaine per 10 lb. bodyweight was used, bodyweight being estimated by guesswork.

Only one observation was possible in most cases, the lignocaine being administered usually to give relief while the owner considered the pros and cons of euthanasia. Duration of the effect could not be checked regularly for the same reason.

All canine choreas seen could be firmly established as the result of a distemper-type virus infection.

Results

Two typical histories are given, and the remainder are set out in the form of a Table.

April, 1957. 5-month-old cross Terrier dog pup, weight about 15 lb. No change in twitches for 2 weeks but classed as "not settled." Left foreleg C, other 3 legs B, unwilling to get up or move about. Twitch rate 60 per minute.

1¼ ml. 10 per cent. lignocaine subcutaneously. Three minutes later B twitches disappeared, C twitches rate up to about 120 per minute but much weaker. Five minutes after the injection all twitches gone.

The dog seemed much happier, walked about freely and easily, but trailed the left foreleg like a radial paralysis.

C twitch returned at about 30 minutes after injection, B twitches at about 90 minutes.

May, 1958. Collie dog pup, 5 months old, 20 lb. Variable cough and diarrhoea for 2 weeks. A storm of epileptiform fits disappeared after 6 hours of pentobarbitone sodium anaesthesia. Subsequently over 48 hours B type chorea appeared in all 4 legs. The type of twitch had to be estimated from its distribution, for the pup refused to rise or take any notice of its surroundings, except to snap whenever it was touched.

2 ml. lignocaine 10 per cent. subcutaneously in the neck.

Over 5 minutes twitches faded out. The expression on the pup's face changed back to the "anxious to please" attitude of an average pup. Drank a little milk, stood up, staggered a little, regained control of its limbs, confidence in its legs returned, and it trotted away to curl up in its favourite spot on the kitchen hearthrug.

From 30 to 120 minutes after the injection the twitches returned in full.

Goldman (1952) reported procaine amide *per os* as a means of controlling Huntington chorea of man in the U.S.A. There was an inexplicable variation in potency from case to case. De Myer & Dyken (1954) and Lazarte *et al.* (1955) were unable to support Goldman's findings.

Procaine amide definitely gives some relief to canine chorea but, from only 2 cases, it seems necessary to give heavy dosing at 3-hourly intervals, which would, in the author's opinion, quickly produce toxic effects on blood cells.

Discussion

Lignocaine cuts out A, B, and C type twitches with only occasional toxic signs; procaine, and procaine amide are less potent. The action is not on the muscles or on motor nerve endings in the muscles. Anaesthesia of the γ fibres in nerve trunks could be responsible—Matthews and Rushworth (1957) found that these γ fibres can be anaesthetised by procaine long before large α fibres. The very tiny doses of lignocaine required to produce a result by intravenous injection, however, and the apparently normal reflexes

(*Concluded at foot of col. 1, page 1206*)

TABLE

Date	Breed	Description of Dog: Age	Sex	Weight lb.		Chorea	Dosage of lignocaine	Results	Notes
March, 1957	Half-bred Alsatian	5 mths.	Dog	15	Settled	B. All 4 legs A. Jaw	3 ml. 3% subcutaneously	All twitches disappeared	Anxious expression lost, walked, previously refused to move.
Sept., 1957	White miniature Poodle	5 mths.	Dog	8	Settled	C. Right foreleg A. Other 3 legs	1 ml. 10% subcutaneously	ditto	Right foreleg left with radial paralysis appearance, vomited. Fit reported.
Sept., 1957	Black and brown cross Terrier	1 yr.	Dog	20	Not settled	B. Jaw A. All 4 legs	1 ml. 3% intravenously	ditto	Twitches returned after 10 minutes.
March, 1957	Black and white mongrel	5 mths.	Dog	12	Not settled	B. Right foreleg	3 ml. 3% subcutaneously	ditto	Anxious expression lost.
Nov., 1957	Mongrel Collie	6 mths.	Dog	25	Not settled	B. All 4 legs	2½ ml. 10% subcutaneously	ditto	Anxious expression gone but too inco-ordinate to stand before and after.

(*Continued on opposite page*)

Date	Breed	Description of Dog: Age	Sex	Weight lb.	Chorea		Dosage of lignocaine	Results	Notes
March, 1958	Collie	1 yr.	Dog	35	Settled	B. Left hind leg A. Right hind leg	3½ ml. 10% subcutaneously	No effect	Inco-ordinate behind before and after injection.
April, 1958	ditto	ditto	ditto	35	ditto	ditto	3½ ml. 10% intrathoracically	All twitches disappeared	ditto
Jan., 1958	Yellow Labrador	9 mths.	Dog	50	Not settled	B. Both hind legs	5 ml. 10% subcutaneously	No effect	Inco-ordinate behind before and after.
Jan., 1958	ditto	ditto	ditto	50	ditto	ditto	8 ml. 10% subcutaneously	Double frequency and half amplitude, of twitches	More inco-ordinate. Vomited.
Jan., 1958	White miniature Poodle	4 mths.	Bitch	8	Not settled	B. All 4 legs	¾ ml. 10% subcutaneously	All twitches disappeared	Gross inco-ordination not changed
April, 1958	Corgi	3 mths.	Dog	7	Not settled	B. Both ears	¾ ml. 10% subcutaneously	No effect	Gross inco-ordination unchanged.
April, 1958	ditto	ditto	ditto	7	ditto	ditto	¾ ml. 10% intrathoracically	All twitches disappeared	ditto
Feb., 1957	Alsatian	10 mths.	Dog	40	Not settled	B. left foreleg B. Both hind legs	12 ml. 3% subcutaneously	No effect	Further observations not possible.
March, 1957	Boxer cross	18 mths	Dog	50	settled	B. Jaw	5 ml. 10% subcutaneously	No effect	ditto
April, 1957	Golden Labrador	1 yr.	Dog	60	settled	A. Jaw	6 ml. 10% subcutaneously	No effect	ditto
June, 1958	Golden Labrador cross	5 mths.	Bitch	30	Not settled	B. Right foreleg	3 ml. 10% subcutaneously	All twitches disappeared	
June, 1958	Cross Terrier	8 mths.	Bitch	20	Not settled	A. Left foreleg	2 ml. 10% intravenously	ditto	
June, 1958	ditto	ditto	ditto	20	ditto	ditto	250 mg. procaine amide *per os.*	All twitches disappeared for 3 hours	
Dec., 1957	West Highland	9 mths.	Dog	10	Not settled	B. All 4 legs and head	1 ml. 10% subcutaneously	All twitches disappeared	Twitches started to return after 20 minutes only.
Dec., 1957	ditto	ditto	ditto	10	ditto	ditto	250 mg. procaine amide *per os.*	All twitches damped down so that dog comfortable. No longer whining. Maximum duration 2 hours.	
Dec., 1957	Collie	6 mths.	Bitch	20	Not settled	A. Abdominal muscles	2 ml. 10% subcutaneously	Twitch disappeared	Completely inco-ordinate before and after dose.
June, 1958	Cross Terrier	8 yrs.	Bitch	25	Normal dogs. Check on general appearance and activity, placing and righting ability, eye, ear twitch, scratch reflex, pulse. 1 ml. 10% given subcutaneously per 10 lb. bodyweight and everything re-checked after 20 minutes.			No effect.	
June, 1958	Golden Cocker	8 yrs.	Dog	35				Vomited, slightly inco-ordinate.	
June, 1958	Collie	5 yrs.	Bitch	30				No effect.	
June, 1958	Collie pup	6 mths.	Bitch	20				No effect.	

Displacement of the Bovine Abomasum

A Clinical Note on Surgical Intervention

BY

E. P. BARRETT and J. NICOL
Guildford, Surrey

IT is only during the last decade that displacement of the bovine abomasum has come to be recognised, and within this period about a dozen authors have contributed to the literature on the subject. From discussions among clinicians it appears that many of those working in the field are each finding a small number of cases. Many members of the veterinary profession at the present time may well be wondering how long this condition in cattle has been occurring: is it an abnormality that has appeared recently as the result of selection for milk production or because of generations of intensive herd management, or has the condition been occurring for a long time and we and our forebears have failed to recognise it? Perhaps these questions may never be answered, but one further uncertainty which may one day be elucidated is that of knowing if the displaced abomasum can pass back to the right side of the rumen spontaneously. Some authors have suggested that such migration does in fact take place, but their evidence has been dependent in large measure upon auscultation of the abdomen. This technique can often defeat even the most experienced observer because adventitious sounds can vary from hour to hour. The true answer to this possible passage from left to right will only be forthcoming when those who have the facilities can carry out some further investigation and furnish positive proof. In the meantime, clinicians working in the field are faced with the problem of knowing how to deal with the ailing cow that has a displaced abomasum.

In our present state of knowledge, the most practical solution appears to be that of returning the viscus to its normal position as quickly as possible. Clinical evidence shows that a really ill animal can quickly return to health when once the abomasum is returned to the right side of the rumen. Several authors have described operative surgical techniques which involve unilateral or bilateral laparotomy. Jennings (1957) described a method in which the abdomen is entered through a single incision in the left sublumbar fossa. Division of the greater omentum is carried out by tearing it near to its rumenal attachment so that the operator is able to make certain that the displaced viscus is left well to the right of the rumen. It is pointed out that the operator's hand can be passed over the top of the rumen, down and underneath the rumen from the right side to reach the abomasum. It is further pointed out that if necessary two operators can enter the abdomen through the same left side incision: one can pass a right hand to the left of the rumen and the other can pass a left hand over the top of the rumen to its right side and the abomasum can then be passed from one operator's hand to the other.

Begg and Whiteford (1956) described a method of casting the patient and attempting to manipulate the abomasum to the correct position although they frankly admitted that this method was not always successful. Gould and Gould (1957) reported success in two cases by casting and rocking the animals.

Because the total number of cases of displaced abomasum seen by individual members of the profession is comparatively small the condition is arousing considerable interest; moreover, because the literature on the subject to date is not extensive, no excuse is felt necessary for recording the technique that was used in the following few cases in which it was possible to compare the results of manipulative surgery with operative surgery.

Case 1: Jersey Cow, 3rd Calver—14 Days Calved.

This animal was cast and manipulation and rolling were carried out without success. It was decided, therefore, to resort to operative surgery, and after using Jennings's method involving two operators the animal made a remarkable recovery.

"Chorea" of the Dog and Lignocaine—*Concluded.*

seen after dosing normal dogs, suggests some kind of central action.

Lignocaine seems to have a remarkable specificity for twitching neuromuscular systems. C type may be visualised as a muscle without a motor supply unaffected by twitches—hence lignocaine suppression of twitching units leaves a paralysed muscle; A and B type muscles have a lesser and a greater proportion of twitching units which, when suppressed by lignocaine, leave a residue of normal functioning muscle.

The compounds discussed here must have a very limited usefulness to a clinician but they would seem to provide a lead for a laboratory neurologist.

Acknowledgments.—Thanks are due for help and advice to the veterinary surgeons of the practice; lay assistant, Mrs. M. Scott; a student, Mr. M. McMurtry; and especially to the veterinary neurologist, Dr. A. C. Palmer. Willows Francis Limited kindly supplied the lignocaine solutions.

References

DE MYER, W., & DYKEN, M. (1954). *Am. J. Med. Sci.* **228.** 70.
GOLDMAN, D. (1952). *Ibid.* **224.** 573.
LAZARTE, J. A., BAARS, C. W., & PEARSON, J. S. (1955). *Ibid.* **229.** 676.
MCGOVERN, V. J., STEEL, J. D., WYKE, B. D., & DODSON, M. E. (1948). *Aust. J. Exp. Biol. and Med. Sci.* **28.** 433.
MATTHEWS & RUSHWORTH. (1957). *J. Physiol.* **135.** 245.
WHITTIER, J. R. (1956). *Am. J. Vet. Res.* **17.** 720.

Case 2: A First Calf Guernsey Heifer 7 Days Calved.
This animal responded immediately and completely to one treatment of casting and rocking.

Case 3: Guernsey Cow, 3rd Calver—10 Days Calved.
Encouraged by the spectacular result obtained in the first calf heifer (Case 2) this animal was cast, rocked and manipulated on 3 separate occasions with no improvement whatsoever, and the animal remained ill. It was decided, therefore, to resort to operative surgery. Laparotomy was carried out in the left flank, and the greater omentum was torn. When, however, the operator passed a hand to one side of the rumen and an assistant to the other it was found that the mesentery interposed as attempts were made to pass the abomasum from one hand to the other. Consequently it was not possible to draw the abomasum well to the right of the rumen and it was therefore pushed down from the left side as far as possible under the rumen. The operation was concluded with no great expectation of a successful result.

The prognosis proved correct and the animal showed no improvement. A letter was sent to Mr. Sydney Jennings at Glasgow University describing the difficulty that was encountered and his reply is thought worthy of repetition. It reads as follows: "When the laparotomy is made in the left sublumbar fossa and the hand is passed over the top of the rumen, any difficulty in defining viscera clearly is probably due to the fact that the hand is to the left of the mesentery. To avoid the mesentery, pass the hand over the top of the rumen and continue moving in the direction of the right hip joint until the right abdominal wall is palpated. Now slide the fingers anteriorly along the abdominal wall until the outline of the liver can be defined clearly, without anything intervening between the hand and the liver. The hand can then be passed downwards and forwards to the omasum and to the ventral and anterior wall of the rumen." With this helpful advice the cow was seen again, the original wound was reopened, and by following the directions it was possible to pass the abomasum from one hand to the other under the rumen and to draw it well up into the right sublumbar fossa. The cow began to improve quickly after the second operation and made a complete recovery.

Case 4: Jersey Cow, 4th Calver—Calved January 3rd, 1958.
This animal went down with milk fever on the day of calving and responded to treatment. There was apparently a slight relapse 6 days later. Although she ate little or no concentrates or silage during the next 3 weeks she was not seen again until early in February when displacement of the abomasum was suspected. Manipulative treatment having proved unsuccessful, once again operative surgery was decided upon. This was undertaken during the period that elapsed between the unsuccessful operation on Case 3 and the receipt of the letter of advice from Mr. Jennings. The same difficulty was encountered as in the first operation on Case 3. Interposition of the mesentery again prevented the passage of the viscus from the operator's to the assistant's hand. The greater omentum having been divided, the abomasum was pushed by the operator under the rumen and as far as possible up the left flank. A doubtful prognosis was given. In this case, however, appetite improved immediately, though very slightly, and although recovery was slow, after 10 days the animal was eating normally. She made subsequently a good recovery of health and milk yield.

Discussion
Everyone would agree that for the return of a displaced abomasum, simple manipulation, if successful, is to be preferred to operative surgery. When the simple procedure fails, however, operative surgery appears to be justified and the tearing of the greater omentum undoubtedly makes it possible to leave the abomasum so well placed to the right of the rumen that its tendency to return to the left is overcome.

It is hoped that the description of the difficulties encountered in 2 of the cases, together with the recounting of the advice which was received and successfully acted upon, will be of service to others who may attempt to use the valuable operation for the return of the displaced abomasum by the use of Jennings's technique.

References

BEGG, H., & WHITEFORD, W. A. (1956). *Vet. Rec.* **68**. 122.
GOULD, A. C., & GOULD, C. M. (1957). *Ibid.* **69**. 516.
JENNINGS, S. (1957). *Ibid.* **69**. 435.

Clinical Note

CYSTICERCOSIS IN AN ORYX

D. C. Taylor
Rochdale

Subject. Ten-year-old male oryx.

History. The oryx had been captive in a zoo for many years. It was discovered dead one day. No signs of ill-health had been previously observed other than mild lameness.

Post-mortem *examination.* On examining the cadaver it was found that the liver had numerous pin-head sized cysticerci scattered over the surface and throughout the substance. There was an acute fibrinous pericarditis with small nodules, 1 to 5 mm. in size containing cysticerci, scattered throughout the myocardium of both sides of the heart. Several cysticerci were lodged as emboli within coronary vessels and there were resultant areas of thrombosis. One minute cysticercus was found on section of the left masseter muscle. Death was presumed to be due to the acute pericarditis and coronary thrombosis. Parasitological examination revealed that the cysticerci were *Cysticercus bovis*, the larval stage of the human tapeworm, *Taenia saginata.* It was considered that infection was less than one year old. Suspected sources of infection were the zoo keepers, and excavations involving lavatories near the stockade containing the oryx.

Acknowledgments.—I would like to thank Dr. W. F. H. Jarrett for his comments on the case and Dr. P. L. Le Roux for the parasitological examination.

Infectious Avian Encephalomyelitis

A Description of the Disease as Seen in England and Wales and an Account of Some Experimental Investigations

BY

L. M. MARKSON

AND

J. D. BLAXLAND

Ministry of Agriculture, Fisheries and Food,
Central Veterinary Laboratory, New Haw,
Weybridge, Surrey.

SUMMARY.—A non-purulent encephalomyelitis of baby chicks in England and Wales is described, clinically and histopathologically indistinguishable from infectious avian encephalomyelitis as seen in the U.S.A. The disease was transmitted by intracerebral and intraperitoneal inoculation of day-old chicks. Attempts at transmission by contact failed. A subclinical encephalomyelitis was demonstrated histologically in some of the progeny of survivors. Subclinical meningoencephalitis was seen in mice and rabbits after intracerebral inoculation with infective chick brain. Epidemiological aspects of the disease are discussed.

Introduction

AT the beginning of 1951 infectious avian encephalomyelitis, already well known in the U.S.A., had not yet been recognised in Britain. In January of that year one of the authors (L.M.M.) began a study of all cases submitted to the Poultry Diagnosis Department at Weybridge, showing symptoms apparently referable to the nervous system. Six weeks after the beginning of this work a non-purulent encephalomyelitis was encountered in young chicks, to be followed intermittently by other outbreaks in which affected chicks showed symptoms and lesions at first suspected and later recognised as those of infectious avian encephalomyelitis, as reported briefly in a preliminary communication (Markson & Blaxland, 1955). Since then well over 200 outbreaks have been recorded and the disease has been studied in the field and the laboratory. This paper is an account of these studies and of experimental investigations.

Symptoms

Although definite symptoms are rarely seen in chicks less than 1 week old, reports from some observant hatchery managers indicate that many baby chicks in a hatch in which the disease is subsequently diagnosed may appear lethargic and abnormally small. The first recognisable sign of the disease is weakness of the legs. Affected chicks are unsteady and often flop on to their hocks. After this stage they have difficulty in standing, move reluctantly when stirred, but prefer to squat. Later they shuffle about on their shanks and ultimately are found lying on their sides helpless with posterior paraplegia. This sequence of events is often accompanied by paralysis of the wings. Fine, rapid, muscular tremors sometimes appear during the later stages of paralysis. They may be so fine as to be appreciated only by holding the affected chicks in the hand; but usually they are clearly visible in the head and neck, or the tail feathers. The appearance of these tremors is not a constant feature, but their absence in the early cases cast some doubt on the identity of the disease. Although many of the chicks which die must do so as the result of damage to vital nerve centres, disturbance of consciousness and of vital functions is not apparently a constant feature. Paralysed chicks are liable to be killed by trampling; but in small groups, and if able to drag themselves to food and water, they often survive.

Survivors, even if reared to maturity, never completely recover from the damage to the nervous system. Ataxia remains to a varying degree, but does not seriously inconvenience the birds, although in a breeding pen it might affect a cockerel's ability to tread the pullets. Nevertheless, two groups of pullets, survivors of an outbreak, mated to healthy cockerels at the Laboratory for experimental purposes, produced a satisfactory number of eggs of average fertility and hatchability.

TABLE I
INCIDENCE

Month	1954	1955	1956	1957	1958
January	2	2	6	13	33
February	—	1	6	12	15
March	—	8	3	9	9
April	—	0	1	3	9
May	—	1	0	1	2
June	—	1	0	1	1
July	1	3	0	4	4
August	1	0	0	0	2
September	4	0	0	2	0
October	1	0	0	2	4
November	0	1	8	11	30
December	6	5	22	17	
Totals	15	22	46	75	
Percentage of total chick batches examined	0·7%	0·9%	1·3%	3·3%	

Epidemiology

The number of outbreaks of infectious avian encephalomyelitis diagnosed at Weybridge since January, 1954, is shown in Table I.

Diagnosis of the outbreaks recorded in Table I was established by histological examination of tissues removed from ailing chicks received alive at the Laboratory (see later). Occasionally diagnosis was possible on dead specimens provided that death had taken place not more than a few hours before removal of the tissue for examination. Most of these outbreaks were investigated by members of the field staff of the Animal Health Division and this account of the epidemiological features of the disease as it has occurred in recent years is based on a study of their reports.

The seasonal incidence of infectious avian encephalomyelitis was quite marked and investigations indicated that in the majority of outbreaks the eggs involved came from pullet flocks reaching maturity and commencing production during the late autumn. With the growth of the broiler industry in this country, a number of hatcheries operate throughout the year; but the majority of chicks in the industry as a whole are still hatched between November and April every year.

During the past 3½ years outbreaks were traced to many of the large hatcheries in the country as well as to smaller firms and breeding flocks supplying chicks and hatchery eggs; but there was no evidence to indicate survival of infection on a farm from one year to the next. Although some of the large hatcheries were involved more than once in a single season, the infection was never traced to the same breeding flock in the same season. There were no reports of the disease originating in the same flock in two consecutive seasons.

The course of an outbreak could usually be predicted with some degree of accuracy. It was rare to find the disease in more than 3 or 4 consecutive hatches: quite commonly infection was confined to a single hatch. Occasionally there were examples of infection either missing one hatch, or perhaps remaining undetected, but occurring in a subsequent hatch.

In the majority of the outbreaks dealt with at Weybridge symptoms were first seen when the chicks were 2 or 3 weeks old; but during 1957 chicks in several outbreaks showed symptoms between the first and second weeks, while, in one series, symptoms were seen and the disease diagnosed in chicks only 2 or 3 days old. Fresh cases seldom developed in an infected batch after the fourth week.

The morbidity varied from less than 5 per cent. to 50 per cent. of the affected batch. Mortality was high among the ailing chicks if they were left with their companions, but, if removed and reared separately so that they could reach food and water without being knocked over by their unaffected pen mates, many of them would survive. As growth and feathering among survivor chicks were uneven and the leg weakness and inco-ordination of gait persisted to a variable degree, it has become customary to recommend destruction of obviously affected chicks although survivors might mature normally in other respects and lay well.

Outbreaks on broiler plants proved difficult to investigate thoroughly because the chicks making up any one batch inevitably originated from several different breeding flocks and, except where distinctive breeds or crosses were involved, it was impossible to obtain full details. Losses amounting to 300 to 400 chicks in lots of from 3,000 to 10,000 were reported but there was nothing to indicate that concentration of stock and the rearing methods on the average broiler plant aggravated the disease, caused more rapid spread of the infection or resulted in higher losses.

In the majority of outbreaks, the infected chicks were traced to a single supplying flock and there was no definite evidence of transmission of the disease during hatching or incubation, or from one infected batch in brooders to others in contact with them. This absence of obvious infectivity was supported by the results of contact experiments in the Laboratory, which are described later.

Histopathology

No gross lesions have ever been attributed to infectious avian encephalomyelitis, and none were seen in the numerous affected chicks studied. Histological examination was at first confined to the brain and spinal cord. Later, following Jones (1934) and Jungherr (1939), sections of pancreas, gizzard and proventriculus were also examined regularly. Other viscera were sampled intermittently to ascertain the distribution of the lesions. Diagnosis is now made routinely on 2 blocks of pancreas and 2 of lumbosacral cord. In the absence of specific lesions the other tissues are sectioned as well.

Tissues were usually fixed in 10 per cent. formol-saline, embedded in paraffin wax in the usual way and sections stained by Ehrlich's haemotoxylin and eosin. A few specimens were fixed in formol-ammonium bromide so that Cajal's gold sublimate method for astrocytes and the Weil-Davenport silver impregnation method for microglia could be applied.

Microscopically the disease appears as a non-purulent encephalomyelitis characterised by vasculitis, perivascular accumulation of lymphocytes, focal microgliosis and neuronal cell degeneration. The first 3 lesions are distributed at random in varying intensity throughout the central nervous system and are apparent in most of the sections. Neuronal cell degeneration is not ubiquitous. In the brain it appears to be confined to the Purkinje cells of the cerebellum, the nuclei of cranial nerves and other nuclei in the brain stem. Purkinje cell degeneration, when it occurs, appears characteristically as clusters of microglial cells surrounding the affected neuron and extending into the molecular layer. Elsewhere, and particularly in the spinal cord where the lesion is frequently seen, neuronal cell degeneration follows a peculiar and characteristic pattern: margination of the nucleus, trigrinolysis with condensation of the Nissl substance at the periphery of the cell, gradual dissolution of the nucleus, and a state of hyaline degeneration progres-

sing to phagocytosis of the effete neuron by microglial cells.

Degeneration of peripheral nerve fibres has been demonstrated and is probably consequent on the destruction of central neurons.

Focal accumulation of cells of the lymphocyte series has been seen in the viscera of many affected chicks, occurring most frequently (about 70 to 80 per cent. of field outbreaks) in the pancreas, often (about 30 to 40 per cent.) in the musculature of the gizzard and proventriculus and less commonly in the spleen, lung, heart, kidney and liver. Three features distinguish these groups of cells: they are almost invariably nodular, except in the gizzard where they are squeezed out between the muscle bundles; they are essentially paravascular in situation; their type cells are larger than normal lymphocytes and are probably lymphoblasts. Once the lesion has been recognised it is readily differentiated from the normal lymphatic tissue of the viscera by its neatly circumscribed, follicular appearance and the size of the component cells. This is most apparent in the pancreas where the normal and abnormal lymphatic tissue are sometimes juxtaposed. In the musculature of the gizzard and proventriculus the lesion might be confused only with lymphatic leucosis which would, however, tend to be diffuse and more severe. The other viscera do not provide reliable diagnostic criteria, partly because they are less commonly affected, while the interpretation of lymphocytic infiltration of the liver is often difficult.

The outbreak in which symptoms were recognised in baby chicks has provided an interesting series of specimens. In chicks 2 or 3 days old changes in the sections of brain and cord are confined to large neurons of the cord, medulla oblongata and mesencephalon—that is, to cells of ectodermal origin. The mesodermal reaction is negligible. Early lymphoblastosis is visible in the pancreas. One chick 8 or 9 days old, which was killed within 24 hours of the appearance of symptoms, shows the full range of lesions in the brain, with affected neurons and early mesodermal reaction in the cord. Another, which died of the disease, shows nothing beyond neuronal cell degeneration in the cord. Four more affected chicks killed when a fortnight old show well-developed, typical lesions.

In one outbreak investigated, the visceral lesions are very pronounced, the encephalomyelitis slight. Transmission experiments with material from this outbreak will be discussed later.

In chickens which survive the acute manifestations of infection, the visceral lesions disappear—they have not been seen in any survivor over 8 weeks of age—while the encephalomyelitis dwindles to a low grade vasculitis and sparse microgliosis which may persist at least as long as 18 months.

As will be discussed later, there is strong circumstantial evidence for the existence in adults of a subclinical carrier state. Such cases have not, however, been examined histologically, as they cannot yet be recognised.

Differential Diagnosis

A detailed discussion of the differential diagnosis of the nervous diseases of poultry is beyond the scope of this paper. In brief, the disease described is clinically and pathologically indistinguishable from epidemic tremor as first reported by Jones (1932 and 1934). Van Roekel *et al.* (1938) rejected the name "epidemic tremor" suggesting instead infectious avian encephalomyelitis as being pathologically descriptive and explicit. As Newcastle disease frequently causes non-purulent encephalomyelitis in poultry, this possibility should be eliminated in field outbreaks suspected as infectious avian encephalomyelitis. It became routine procedure during the course of these investigations to test blood samples from suspected cases of infectious avian encephalomyelitis for evidence of haemagglutination inhibiting antibodies for Newcastle disease and all outbreaks in which the clinical or epidemiological features were at all suspicious were investigated by the field staff of the Animal Health Division.

Experimental Work

Inoculation of Chicks with Crude Brain Suspension

The disease has been transmitted many times by intracerebral inoculation of day-old chicks with a suspension of brain from field cases in distilled water, 0.85 per cent. saline or 10 per cent. guinea-pig serum-saline. Day-old chicks were used because the paper-thin cranium could be penetrated easily with a fine needle and the amount of tissue suspension inoculated was 0.03 ml.—the maximum amount which could be introduced with safety. The disease was usually reproduced in about 90 per cent. of chicks which survived inoculation and death from other causes. The entire range of symptoms was seen in inoculated chicks after an incubation period of from 2 to 4 (exceptionally 7) weeks. The encephalomyelitis was reproduced, although with greater intensity than in field cases, but the visceral lesions were rarely present. The same results were obtained after intraperitoneal inoculation.

Early in this work control inoculations were made using normal saline alone and suspensions of brain from healthy chicks. Neither symptoms nor lesions were seen in these experiments.

Numerous attempts to isolate bacteria from field cases and from the experimental inocula have failed.

Variations in virulence have been seen among the different strains of the infective agent. Thus in several transmission experiments intracerebral inoculation of chicks failed to provide symptoms, although slight to moderate lesions were demonstrated in the central nervous system. The possibility of the experimental chicks being immune was not a likely explanation as they were produced by the Laboratory breeding flock in which there has never been any reason to suspect the existence of this disease. Material from the outbreak already mentioned, in which the visceral lesions predominated, yielded interesting results when tested for transmissibility. The usual brain suspension was prepared and inoculated into day-old chicks by the

PLATE I.—An affected chick in characteristic attitude, squatting on its shanks but with appetite undiminished.

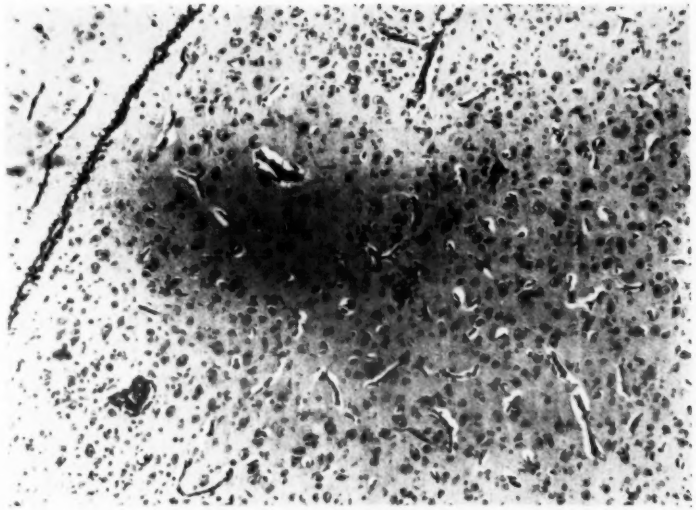


PLATE II.—Cerebrum of an affected chick. A few perivascular lymphocytes and scattered microglial cells. × 60.

The photomicrographs are all from sections stained by haematoxylin and eosin.

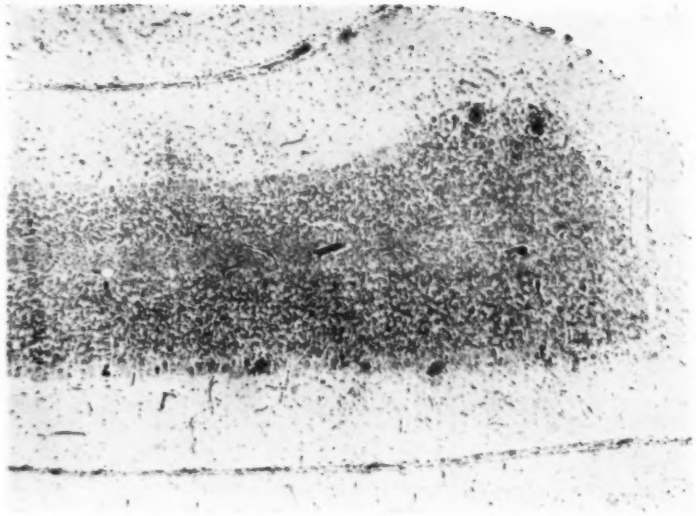


PLATE III.—Cerebellum of an affected chick. Characteristic reaction at the *stratum Purkinje*, microgliosis in the *stratum molecularis* and perivascular lymphocytes in the white matter. × 60.

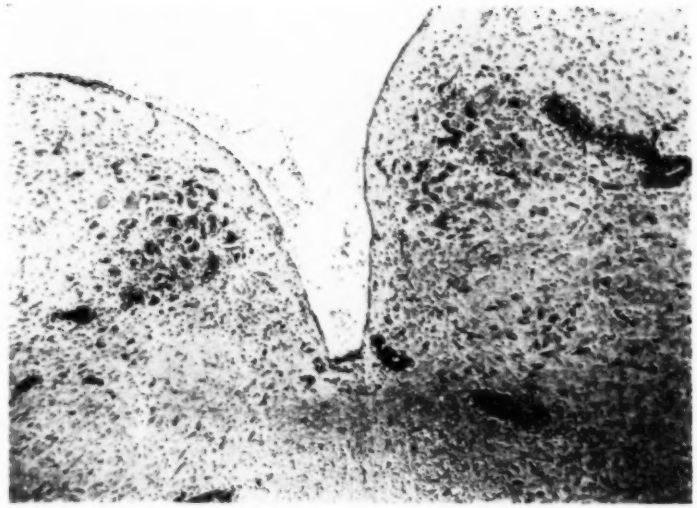


PLATE IV.—Medulla oblongata of an affected chick. Severe inflammation of the vagal and hypoglossal nuclei. × 60.

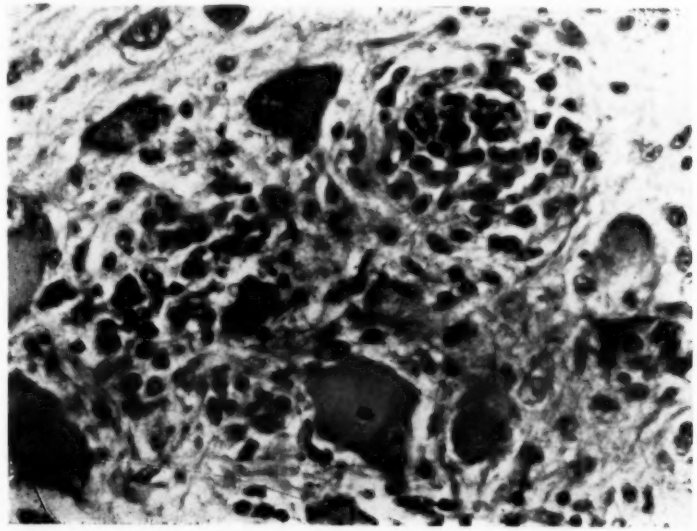


PLATE V.—Spinal cord of an affected chick. Microgliosis and neuronal cell degeneration. × 510.

PLATE V.—Spinal cord of an affected chick. Microgliosis and neuronal cell degeneration. × 510.

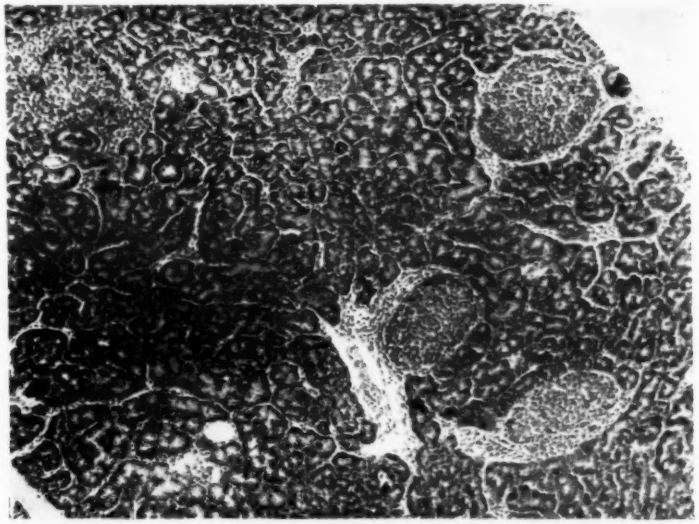


PLATE VI.—Pancreas of an affected chick. A typical field. × 115.

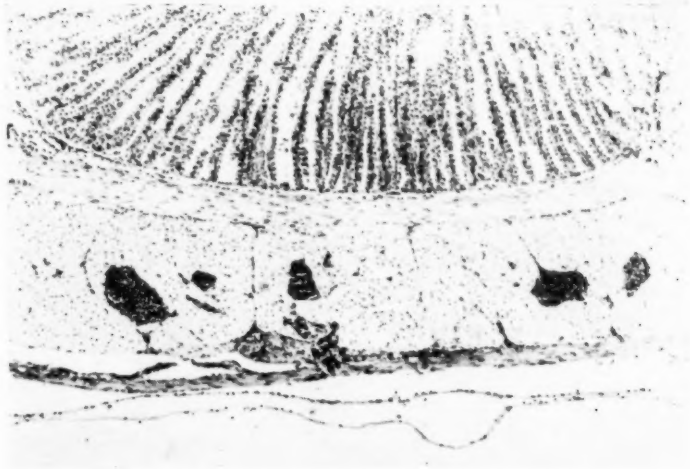


PLATE VII.—Proventriculus of an affected chick. The paravascular nodules of lymphoblasts are irregular in shape probably because of muscular pressure. × 70.

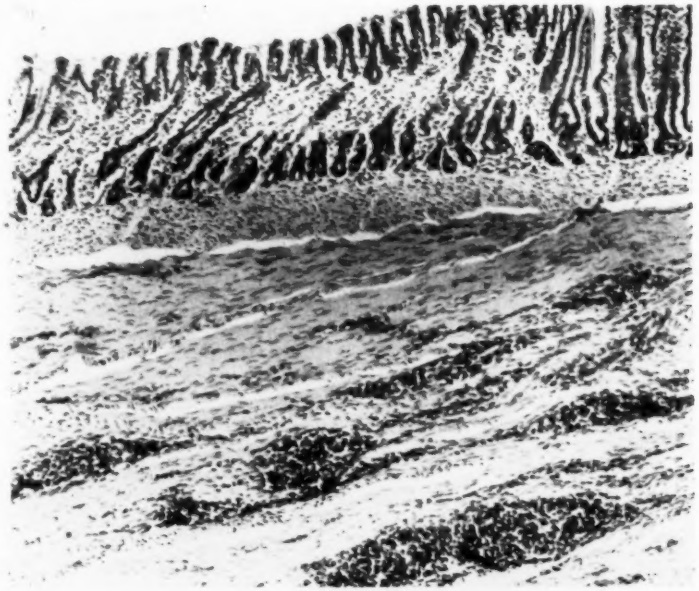


PLATE VIII.—Gizzard of an affected chick. The lymphoblastosis, although essentially paravascular, is beginning to extend along clefts between the bundles of muscle fibres. × 125.

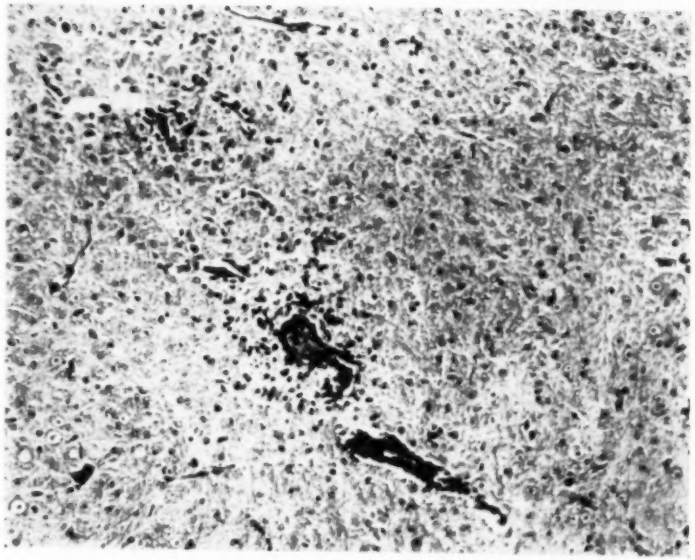


PLATE IX.—Thalamus of an inoculated mouse. Non-purulent inflammation. × 130.

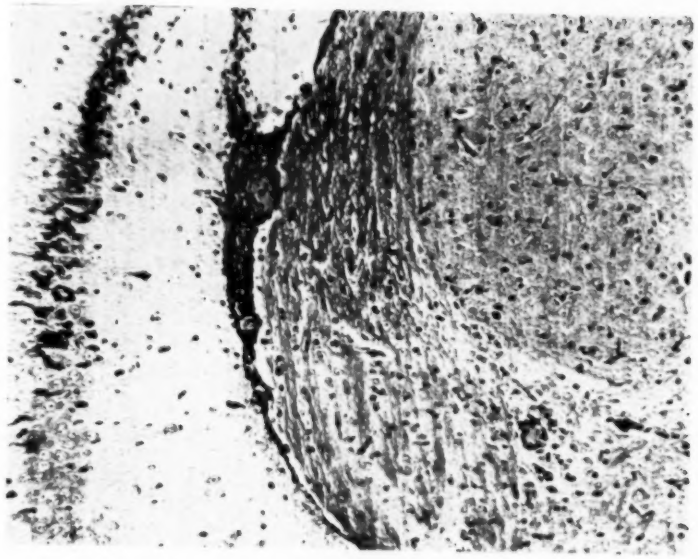


PLATE X.—Brain of an inoculated mouse. Non-purulent meningitis. × 130.

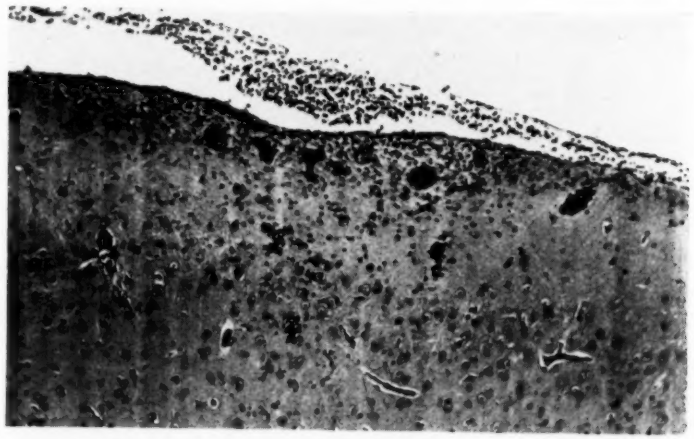


PLATE XI.—Brain of an inoculated rabbit. Non-purulent meningo-encephalitis. × 80.

intraperitoneal or intracerebral routes. Those inoculated intraperitoneally survived apparently in good health for 8 weeks, when they were killed and examined. Encephalomyelitis was minimal or absent in representative sections, but nodular lymphoblastosis was seen in all the spleens and several other viscera. The chicks inoculated intracerebrally were killed as symptoms appeared, which occurred in all but one. No lesions were found in sections from this survivor, but all the others showed moderate to severe encephalomyelitis. Visceral lesions were found in 4 out of 5 chicks which lived longer than 32 days and in the lungs of one of them.

Inoculation of Chicks with Cell-free Material

Although infectious avian encephalomyelitis is recognised in the U.S.A. as a viral disease and the histological pattern typifies response of the central nervous system to infection with a virus, confirmation of the aetiology of the British disease was sought by inoculating day-old chicks intracerebrally with a cell-free preparation of infected chick brain.

In the first experiment a passage strain was suspended in the usual way, centrifugalised for 1 hour at 10,000 r.p.m. and the supernatant fluid injected intracerebrally into day-old chicks. Control chicks were inoculated with uncentrifugalised suspension. The chicks were killed at 6 weeks in apparent health and sections of brain examined. Histological evidence of infection was found in 5/14 control chicks and in 2/15 chicks inoculated with the cell-free material.

In the second experiment brain from a field case was used. This time the 10^{-1} suspension was passed through a Seitz EK filter, control chicks being inoculated with unfiltered suspension. Symptoms were first seen in both groups 4 weeks after inoculation. The clinical manifestations of the disease having been established, the chicks were killed during their sixth week and their brains sectioned for histological diagnosis. Lesions were found which confirmed successful transmission in 7/21 of those chicks given the Seitz filtrate and in 8/13 of the control chicks.

Transmission by Contact

Twenty day-old chicks put in the same brooder as 20 chicks inoculated with a field strain of infectious avian encephalomyelitis virus and reared with them until they were 8 weeks old showed neither symptoms nor lesions of the disease. Twelve/20 of the inoculated chicks developed symptoms of infectious avian encephalomyelitis and typical lesions were found in sections of brain and spinal cord of 18/20. Twenty uninoculated chicks were also brooded from day-old with 12 affected chicks obtained from a field outbreak. The latter were 2½ weeks old when received and all died naturally, or were killed during the next 3 weeks. The in-contact chicks were left in the same brooder until they were 8 weeks old without developing either symptoms or lesions of the disease.

Inoculation of Embryonated Chicken Eggs

Twelve-day-old chick embryos were inoculated into the yolk or amniotic sac with 0.1 ml. of 10^{-1} suspensions of infected brain. Hatchability was half that of embryos inoculated with a suspension of normal brain. Half the chicks were killed on hatching, the rest reared to 4 weeks. Sections of brain were prepared from every chick but histological examination revealed no abnormalities.

The Possibility of Natural Transmission through the Egg

The possibility of transmission of the disease through the eggs of pullets which had survived the natural disease as chicks was investigated. Fifty-four affected chicks were purchased from 2 farms, after infectious avian encephalomyelitis had been diagnosed, and reared at the Laboratory. The majority, although ataxic in varying degree, survived to maturity, when 12 pullets were selected and mated to a normal cockerel. Over a 10-month period 250 fertile eggs were incubated. None of the 135 chicks which hatched developed symptoms of infectious avian encephalomyelitis before they were killed for histological examination at 4 to 5 weeks of age. In spite of the absence of clinical disease sections from 12 (8.9 per cent.) showed slight to moderate, non-purulent encephalitis.

A similar experiment was carried out the following year with 12 ataxic pullets, survivors of another outbreak. Over an 11-month period 1,029 eggs were incubated, of which 881 were fertile. Six hundred and thirteen chicks hatched of which 43 died from non-specific causes and 129 were used for another experiment. Four hundred and forty-one were reared for from 5 to 6 weeks when they were killed for histological examination. One of these was paraplegic on hatching and showed degenerating neurons in the spinal cord. Slight to moderate, non-purulent encephalomyelitis was demonstrated in 171 (38.8 per cent.) of the remaining 440.

Portions of brain from chicks showing lesions of encephalomyelitis are being held at $-20°$ C. to be tested by chick inoculation for the presence of the virus.

Susceptibility of Small Laboratory Mammals

Infected chick brain was inoculated by the intracerebral route into young mice, guinea-pigs and rabbits, with inconclusive results. In the first experiment 6 mice, 4 rabbits and 4 guinea-pigs were inoculated with a 10^{-1} suspension of chick brain from a field case of infectious avian encephalomyelitis, receiving 0.04, 0.4 and 0.2 ml. respectively. A group of day-old chicks, inoculated at the same time with the same inoculum, acted as controls by demonstrating the virulence of the material. The mammals were killed in apparent health 4 weeks after being inoculated. Sample sections of their brains contained no evidence of disease. The experiment was subsequently repeated with the intention of confirming this insusceptibility to the virus. Six mice, 2 rabbits and 4 guinea-pigs were inoculated by the same method as before, with chick brain obtained from another outbreak of the disease. This strain, too, was pathogenic for chicks following intracerebral inoculation. One of the rabbits did not recover

from the anaesthetic, and 2 of the mice died for no demonstrable reason. The remaining animals seemed quite healthy when they were killed 4 weeks after inoculation. The brains were removed immediately and sections prepared. Those from the guinea-pigs were normal; those from the 4 mice and the rabbit which had survived to the end of the experiment presented a non-purulent meningo-encephalitis. The reaction did not follow in every detail the pattern seen in chicks. Microgliosis was locally fairly intense and vasculitis marked, but perivascular cuffing with lymphocytes was less pronounced and the characteristic degeneration of large neurons was absent. The presence of meningitis was quite unlike the tissue reaction in the chick in which host this feature had not yet been seen. Photomicrographs illustrating this meningo-encephalitis are attached. As the virus of infectious avian encephalomyelitis has invariably been reported non-pathogenic for mammals and as the first experiment had appeared to confirm this, no tissue was harvested in the second experiment for passage back into chicks. The experiment was therefore repeated on 12 mice, 4 rabbits and 6 guinea-pigs, portions of brain from each animal being stored at −20° C. until the sections had been examined. The brain of 1 mouse showed evidence of slight encephalitis. In 2 of the rabbits non-purulent encephalitis was moderate to severe. Passage from the rabbits back into chicks resulted in occasional, slight, symptomless encephalitis. Attempts at further passage in rabbits were completely negative.

Non-purulent encephalitis has not been encountered before in the small mammal stock at this Laboratory. This pathogenecity of infectious avian encephalomyelitis virus for mice and rabbits warrants further investigation.

Discussion

The disease described in this paper is clinically and pathologically indistinguishable from infectious avian encephalomyelitis and the results of the experimental work and epidemiological investigations support most but not all of the findings of the American workers who have studied the disease.

Experimental transmission of the disease by inoculation of chicks with cell free filtrate of infected brain as well as with crude unfiltered suspension confirmed the viral aetiology of the disease as reported by Jones (1934) and Van Roekel *et al.* (1938). Jungherr (1939) did not find visceral lesions in his experimentally infected chicks, whereas the work presented in this paper includes a record of one strain of virus which possessed marked viscerotropism and minimal neurotropism.

The published reports concerning egg inoculation experiments are conflicting. Jones (1936), Kligler and Olitsky (1940) and Feibel *et al.* (1952) all agreed that the chicken embryo was not apparently susceptible to the virus. On the other hand, Van Roekel *et al.* (1941) demonstrated infectious avian encephalomyelitis in chicks hatching from eggs which had been inoculated before incubation or when the embryos were 10 to 11 days old. Jungherr and Minard (1941) obtained similar results following intravenous and intracerebral inoculation of embryos. Jungherr, Sumner and Luginbuhl (1957) reported successful adaptation of infectious avian encephalomyelitis virus to chicken eggs by intraocular inoculation of the embryo. Wills and Moulthrop (1956) used this technique successfully and also infected chick embryos with field virus inoculated *via* the yolk sac. There therefore seems no doubt that chicken embryos can be infected with this virus and, in spite of the negative results obtained in the small experiment reported here, extensive egg inoculation experiments to explore this point further did not seem warranted.

The experiments reported in this paper support the impression gained from a study of field outbreaks of infectious avian encephalomyelitis that there is no danger of transmission of this disease through contact with infected chicks during rearing and this seems to be the general opinion of the American workers. The only contrary report from the U.S.A. is that of Van Roekel, Bullis and Clarke (1939). More recently the 1949 Annual Report of the Animal Research Division of the New Zealand Department of Agriculture reported the first outbreak of infectious avian encephalomyelitis in that country. Here again disease was apparent in chicks of only 1 of 2 breeds, both being reared in the same brooder house. There therefore seems little likelihood of spread of this disease through hatchery or brooder contact.

Several groups of American workers have sought, without success, to demonstrate infectious avian encephalomyelitis in the progeny of survivors of a previous outbreak (Jones, 1934; Bottorf *et al.*, 1936; Schaaf & Lamoreux, 1955); but these workers all accepted symptoms as the criterion of infection. In the experiments described here, non-purulent encephalomyelitis was demonstrated histologically in the progeny of 2 separate groups of survivors, although all but 1 of the chicks had been reared for 5 to 6 weeks without showing symptoms of infectious avian encephalomyelitis.

The demonstration of encephalomyelitis among some of the progeny of survivors which have reached maturity could be taken as evidence that the survivors of this disease or even some of their progeny can act as reservoirs of infection and as such represent a source of danger to susceptible laying pullets. The epidemiological evidence, however, never suggested further spread of the disease in this manner. Although conclusive proof of the egg transmission of infectious avian encephalomyelitis is lacking both in Britain and the U.S.A., the circumstantial evidence in the outbreaks reported in this paper supports the contention of Taylor, Lowry and Raggi (1955) that disease in chicks is due to a transient, subclinical infection in the laying flock during which the eggs may become infected. The fact that it was rare to find the disease in more than 2 or 3 consecutive hatches supports this opinion that the infection in the adult fowl is transient. The significant pause in egg production and temporary decrease in hatchability of eggs, which, according to Taylor *et al.*, are the only symptoms of infection in the breeding flock, were not observed by field

officers investigating the outbreaks under review; but this lack of corroborative evidence might have been due to absence of detailed records on the farms concerned—the outbreak described by the American team occurred in the University flock during a period when eggs were being collected and recorded for experimental incubation.

Recent reports from America have suggested a relationship between lenticular opacity and infectious avian encephalomyelitis (Peckham, 1957; Bridges & Flowers, 1958). An apparently identical ocular disease occurred among 2 groups of growing chickens, all survivors of batches in which infectious avian encephalomyelitis had previously been diagnosed at Weybridge. Similar lesions also developed in one pullet among the first group of survivor chicks reared at the Laboratory for the breeding experiments. On the other hand, cataract reaching epidemic proportions was seen in 1956 in 2 flocks, without previous evidence of infectious avian encephalomyelitis. Outbreaks of cataract, not apparently related to infectious avian encephalomyelitis have also been reported in England by Gordon (1958), in France by Brion (1958) and in the U.S.A. by Flowers *et al.* (1958). It therefore appears that the relationship between the 2 diseases could be coincidental.

Acknowledgments.—The authors are indebted to numerous colleagues in the Animal Health Division of the Ministry of Agriculture, Fisheries and Food for the detailed investigation of field outbreaks of infectious avian encephalomyelitis. The many histological sections examined in the course of this work were prepared by technicians of the Pathology Department under the direction of Mr. N. C. Lacey, F.I.M.L.T.

References

Anim. Res. Div. N.Z. Dep. Agric. Ann. Rep. 1949–50, page 6.
BOTTORF, C. A., TEPPER, A. E., MARTIN, C. L., CHARLES, T. B., & REED, F. D. (1936). New Hampshire Agric. Exp. Sta. Circ. 51.
BRIDGES, C. H., & FLOWERS, A. I. (1958). *J. Amer. vet. med. Ass.* **132.** 79.
BRION, A. (1958). Personal communication.
FEIBEL, F., HELMBOLDT, C. F., JUNGHERR, E. L., & CARSON, J. R. (1952). *Amer. J. vet. Res.* **13.** 260.
FLOWERS, A. I., GRUMBLES, L. C., DU BOSE, R. P., & DELAPLANE, J. P. (1958). *Poult. Sci.* **37.** 420.
GORDON, R. F. (1958). Personal communication.
JONES, E. E. (1932). *Science.* **76.** 331.
———. (1934). *J. exp. Med.* **59.** 781.
JUNGHERR, E. L. (1939). *Poult. Sci.* **18.** 406.
———, & MINARD, E. L. (1941). *J. Amer. vet. med. Ass.* **99.** 215.
———, SUMNER, F., & LUGINBUHL, R. E. (1956). *Science,* **124.** 80.
KLIGLER, I. J., & OLITSKY, P. K. (1940). *Proc. Soc. Exp. Biol. N.Y.* **43.** 680.
MARKSON, L. M., & BLAXLAND, J. D. (1955). *Vet. Rec.* **67.** 131.
PECKHAM, M. C. (1957). *Avian Dis.* **1.** 247.
ROEKEL, H. VAN, BULLIS, K. L., & CLARKE, M. K. (1938). *J. Amer. vet. med. Ass.* **93.** 372.
———, ———, & ———, (1941). *Ibid.* **99.** 220.
SCHAAF, K., & LAMOREUX, W. F. (1955). *Amer. J. vet. Res.* **16.** 627.
SUMNER, F. W., JUNGHERR, E. L., & LUGINBUHL, R. E. (1957). *Ibid.* **18.** 717, 720.
TAYLOR, W. L., LOWRY, D. C., & RAGGI, L. G. (1955). *Poult. Sci.* **34.** 1,036.
WILLS, F. K., & MOULTHROP, I. M. (1956). *Southwest Vet.* **10.** 39.

Current Literature

ABSTRACTS

Successful Closure of a Patent Ductus Arteriosus in the Dog. Report of a Case. SCHMUTZER, KARL J., MARABLE, SAMUEL A., & MALONEY JR., JAMES V. (1958). *Cornell Vet.* **48.** 4. 404.

The subject was a 2-year-old Cocker Spaniel bitch, referred to the authors with the diagnosis of congenital heart disease. Examination showed it to be physically retarded and showing marked tachypnoea. The resting pulse varied between 180 to 200 per minute. Auscultation revealed a harsh, continuous murmur with systolic accentuation, which was maximal between the 2nd and 4th left intercostal space. Electrocardiogram was normal, but phonocardiogram confirmed the auscultatory findings.

A retrograde aortogram was obtained following cardiac catheterisation, and showed immediate visualisation of the pulmonary vascular tree, confirming the presence of a left-right shunt.

The defect was subsequently exposed by thoracotomy through the left 4 intercostal space, and once dissected free, the patent ductus was closed by purse-string sutures at the aortic and pulmonary junctions, with 2 transfixion sutures in between. Recovery was complete and uneventful.

R. G. W.

An Outbreak of Haemorrhagic Pneumonia in Mink. FARRELL, R. K., LEADER, R. W., & GORHAM, J. R. (1958). *Cornell Vet.* **48** (4). 378-86.

In view of the increasing interest in mink farming, the authors' description of an outbreak of haemorrhagic pneumonia in a mink farm is of extreme value to practitioners in this country.

Pneumonia in the mink is sporadic in occurrence being attributed to non-specific causes such as inclement weather, etc. However, in the outbreak described, and in a Danish outbreak described by Knox, *Pseudomonas aeruginosa* was isolated as the cause of the enzootic infections.

The symptoms described are those of shallow, rapid breathing accompanied by the expulsion of a frothy nasal discharge. Death occurred within 24 hours. *Post-mortem* examination showed a bilateral pneumonia. Some of the lung lobes showed intense oedema and congestion, while others showed marked consolidation. Bacteriological cultures revealed the presence of *P. aeruginosa* in most of the body organs including the lungs and pleura. In this particular outbreak the organism was also isolated from the water pots.

Treatment consisted of a general sanitation programme including pen cleansing and disinfection, and the addition of dilute hypochlorite solution to each water pot. Sulphathiazole at the rate of 0.5 g. per pound bodyweight was added to the diet, and each animal received 50 mg. of streptomycin twice daily. As no untreated animals were available, it was impossible to determine whether the drug treatment, sanitation, diminution of susceptibles, or a combination thereof was the deciding factor in the control of the disease.

J. S. J. L.

The New Field Station of the Royal Veterinary College

AS our readers may know, the Royal Veterinary College field station had been established for a number of years at Streatley in Berkshire. Recently the College transferred this part of its activities to a new field station which is located between Potters Bar and North Mimms on the Middlesex/Hertfordshire border. A substantial amount of work had to be undertaken to provide the necessary facilities by way of buildings and equipment, and work on the new field station was divided into three stages, of which two have now been largely accomplished and the third will be undertaken as soon as funds permit. Very substantial grants from the University Grants Committee transmitted through the medium of the Court of the University of London, have enabled the College to purchase the valuable site and to erect the new buildings.

In selecting a new field station, an overriding consideration was the advantage which would result from the comparative nearness to the parent College, the amenities of the estate, and the number of practices from which clinical and pathological material might be obtained. In this latter connexion it is proposed, as soon as is practicable, to invite veterinary surgeons who are within reasonable motoring distance to inspect the field station with a view to enlisting their interest in it.

The field station comprises Hawkshead House and its estate, together with the neighbouring Bolton's Park Farm. There are about 430 acres in all, which should provide ample space for the practical activities of the farm, the study of animal husbandry, and grazing for experimental animals and in-patients.

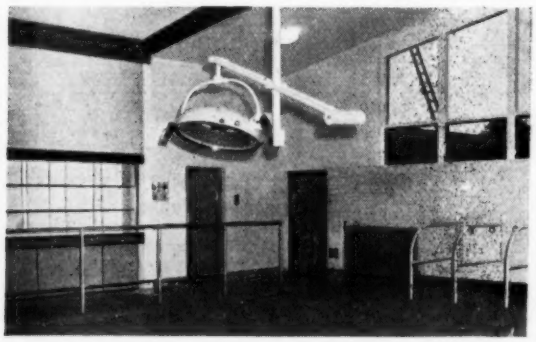
Of the three stages to which reference has been made above, those now largely completed are the clinical units of medicine and surgery. The third and final stage will include provision for pathology —which is at present housed in a converted building —physiology, an obstetric unit, more animal accommodation, and a hostel for 60 students. At present the students are accommodated partly at Hawkshead House, partly in the farmhouse, and otherwise in local lodgings.

For the permanent buildings so far erected a functional but agreeable style of contemporary one-storey architecture with shallow pitched roofs has been chosen. The principal lecture room and the large teaching laboratory are admirably spacious, airy, and light, and provide enviable amenities for the students and their teachers. The common rooms for students and staff, the library, dining hall, refectory, and so on, are situated at Hawkshead House and exemplify the pleasant rooms of a conventional country house.

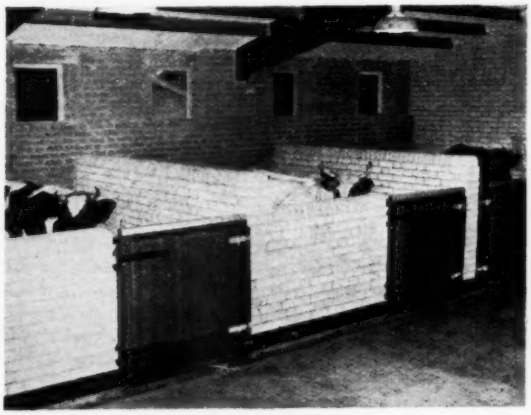
A section of the main teaching laboratory.

Among the completed buildings is a remarkably compact and excellently equipped surgical block. This comprises a large and beautifully lit operating theatre, preparation room, patients' recovery room, X-ray room, and sterilising unit. There are also modern bath and changing rooms for both students and surgeons. Additional surgery accommodation takes the form of what is described as an "operation barn" where patients, which for one reason or another are not suited to the operating theatre, may be dealt with. In the operating theatre proper, in the "barn," and also in the animal houses are ingenious combinations of restraining devices easily adaptable to the various species and to the numbers

A view of part of the main new buildings, including the entrance.



The operating theatre.



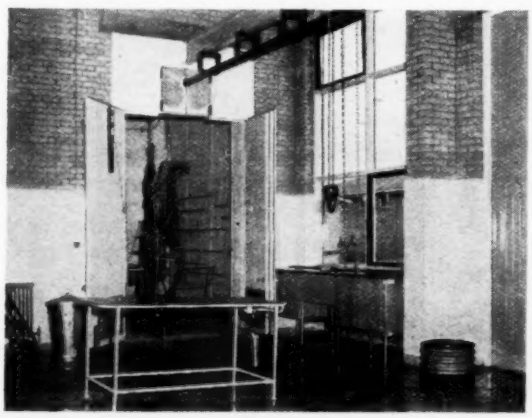
One of the animal houses in which the pens are arranged around three sides of a square.

which may be under treatment or demonstration at any one time.

So far as the clinical work of the field station is concerned, we are asked to appeal to local veterinary surgeons to help in providing material. It having been decided that the station would no longer maintain its own veterinary practice—so as not to embarrass private practitioners nearby—it is necessary to ask such practitioners to make good the deficiency so far as they are able.

The unit devoted to veterinary medicine is comparable in its amenities and modern design with that for surgery. Of note are some of the animal houses in which pens are arranged round three sides of a square, leaving a reasonably sized space in the centre for the students and the members of the academic staff who are addressing them. In this unit, too, is found an ingenious system of heating by electric lamps so that work may be carried on in a reasonable degree of comfort.

A spacious *post-mortem* room is provided with the necessary apparatus and amenities for lectures and demonstrations in meat inspection. A modern incinerator completes this section, and a well-equipped pharmacy has been provided adjacent to it.



The *post-mortem* room, which is also used for demonstrations in meat inspection, showing, in the background, the cooling chamber.

It is understood that the Department of Medicine may need further animal accommodation, as clinical material increases in volume.

Having regard to the comparatively short time since the site was decided upon, the Royal Veterinary College is to be congratulated on the speed with which work has proceeded, the accomplishment to date, and the pleasant appearance and first-rate equipment of that substantial part of the whole premises now in use. Later, when stage III has been completed, this new field station will be a credit to those who have designed it, and an important adjunct to veterinary education in this country.

THE FARMERS' CLUB CUP

The Farmers' Club Cup, awarded annually to the author of the best paper given at the club's monthly meetings, has been won this year by Mr. G. N. Gould, J.P., M.R.C.V.S. The title of his paper, given on February 12th, 1958, was "Farming Methods and Practices and their Relation to Animal Health."

The cup was presented to Mr. Gould, by Lady Cockcroft, on the occasion of the club's annual dinner at Grosvenor House on December 9th. It is the gift of a member of the club who wishes to remain anonymous. The first award was made last year to Mr. Stephen Cheveley, O.B.E., for his paper on "Farm Capital Investment—how will the Government Proposals Work?"

THE REGISTER OF VETERINARY SURGEONS

Admissions to the Commonwealth List. Under the provisions of Section 13 (1) of the Veterinary Surgeons Act, 1881, the names of the undermentioned gentlemen were entered on the Commonwealth List on the dates indicated: COUGHLAN, Richard Francis Ray, D.V.M. (TEXAS), B.V.SC. (SYDNEY), Veterinary Research Institute, Ipoh, Malaya, November 26th, 1958, and SORENSEN, Norman Norgaard, B.V.SC. (SYDNEY), c/o New Zealand House, Strand, London, W.C.2, December 3rd, 1958.

Removals from the Register. Under the provisions of Section 25 (3) of the Veterinary Surgeons Act, 1948, and Statutory Instrument No. 1729 of 1957, the names of the undermentioned gentlemen were removed, at their request, from the Register on December 4th, 1958: LAKE, Herbert Arnold, Daniel's House, Beaminster, Dorset, and PERRIN, Charles William, Avenue House, Cornwallis Avenue, Folkestone, Kent.

CATALOGUE OF VETERINARY FILMS

The Permanent Committee for International Veterinary Congresses hopes to make available early in 1959 its definitive catalogue of films. The price will be about 10s. per copy, and those interested are asked to communicate with Colonel G. V. Short, at Imperial Chemical Industries Ltd., Fulshaw Hall, Wilmslow, Cheshire, indicating the number of copies they are likely to require. No money should be sent at this stage.

News and Comment

VISITORS FROM OUTER MONGOLIA

An unique occasion was provided this term by the visit, on invitation, of two veterinarians from Outer Mongolia to the Cambridge University School of Veterinary Medicine. They are said to be the first persons from that country to visit Britain. They were Professor Badwyn Jarinpil and Mr. Bazarsad, both of the National University of Ulan Bator, the former being its Vice-Principal. At the beginning of their week's visit they were received by the Vice-Chancellor, Lord Adrian. Most of their time was spent at the School, but they also visited the Animal Research Station of the A.R.C., the Cambridge Cattle Breeders' A.I. Centre, the Veterinary Investigation Centre, the Equine Research Station at Newmarket, and one of Messrs. Chivers' farms. The Chief Veterinary Officer of the Ministry of Agriculture arranged for them to visit the headquarters of the Animal Health Division at Tolworth, and the laboratories at Weybridge after they left Cambridge, and they also visited the laboratories of Messrs. Glaxo and Allen & Hanbury.

The Mongolian People's Republic

The country from which these visitors came is a mountainous tract with plateaux of an average height of 4,500 feet above sea-level bounded on the north by the U.S.S.R. and on the south, east, and west by China. For most of the year the cold is intense, the yearly average temperature in the north being below zero C. The average annual rainfall for the whole country is under 10 inches.

Many fossils of prehistoric animals are found in parts of the country, which has a long history, distinguished in particular by the exploits of Ghengiz Khan. Primitive conditions persisted until about 1921 when the events leading to the establishment of the Mongolian People's Republic as it now is, took place. Since then, with Russian assistance, there has been a great drive to introduce modern civilisation.

Veterinary Services

Most of the people still remain nomadic and the main occupation is animal husbandry. The development of veterinary services has been rapid and remarkable. From practically nothing an elaborate organisation has arisen. There are now veterinary surgeons in every regional capital and State farm, and in various outside areas, with "veterinary assistants" in every village. Motorised units visit other areas. Training was at first carried out in Russia, but in 1942 the National University was founded with veterinary science as one of its three faculties, the other two being medicine and an omnibus faculty covering everything else. Further faculties have since been added and other universities founded. Much stress is laid on vaccination, and several "biological institutes" now exist for the production of vaccines and antisera. Claims are made for considerable progress in the elimination of most of the serious animal diseases.

Animal Husbandry

The country, which has an area of some 600,000 square miles and a population of about 1,000,000, is said to have some 26,000,000 animals—horses and sheep in great number, cows (including the yak and a yak-cow hybrid called "hainik"), goats, and camels. These five species, on which the prosperity of the country depends, appear on its emblem. Pigs and poultry are not generally kept except on the State farms where the former were introduced from Russia and the latter from China. Horses play an important rôle in Mongolian life and horse-racing is a popular sport, the races generally being over 20 miles and the horses being ridden by children! The Mongolians milk most of their animals. Mares are said to yield up to 50 gallons in 3 months' lactation. A camel will produce 60 to 70 gallons. The cows are said to give about 150 gallons and some over 200 gallons. Goats give 10 to 15 gallons and ewes 6 to 7 gallons. Sheep constitute 50 per cent. of the animal population and wool is the most important export.

Mongolians have been interested in animal breeding for centuries. Long experience seems to have given them considerable skill in adapting this to local conditions and in producing strains with the necessary stamina for this severe climate and best suited, at the same time, to fulfil the various purposes for which the animals are required.

The Future

Having made such headway, but still being very largely out of touch with the outside world, the leaders of the Mongolian People's Republic are anxious to make contact with other nations and to share in the advances being made elsewhere. Since veterinary science holds such an important place in their economy it was natural that one of the first contacts to be made should be in this field. The visit to Cambridge was a beginning which it is hoped will lead to further contacts of mutual advantage.

Professor W. I. B. Beveridge (centre) with Professor Badwyn Jarinpil (left) and Mr. Bazarsad, veterinarians, of the Mongolian National University, Ulan Bator.
Photograph by courtesy of the Cambridge Daily News.

THE BRITISH AGRICULTURAL HISTORY SOCIETY

The society held its winter meeting in co-operation with the Association of Agriculture, at the Institute of Education, University of London. The morning was devoted to a paper by Dr. F. N. L. Poynter of the Wellcome Historical Library on "The Place of Gervase Markham in English Veterinary Literature." Markham has been breathed upon ever since the Rev. Walter Harte dubbed him the first hackney writer, and Sir Frederick Smith, in his great bibliography, treated him to an excess of abuse, rather undeserved from a cavalry officer whose horses often enough suffered from an excess of Stockholm tar. Dr. Poynter was at pains to vindicate Markham. He was descended from an outstanding family having great connexions. He was one of the foremost riders of his day, and had learnt in the Italian school of Grisone, who was a soldier and something of a poet and dramatist as well as a technical writer. The matter in his "farriery" books was not in advance of the thought of his day, but contains a good deal of protest against some of the cruelties then practised, both in training horses and treating their distempers. Dr. Poynter did not mention the barbarous methods of shoeing then very common. He did, however, restore Markham to the place his voluminous and useful writings deserve, and which the innumerable reprints made throughout centuries up to the 19th certainly support despite the massive condemnation of Sir Frederick.

At another session, Dr. J. K. S. St. Joseph, Curator in Air Photography, University of Cambridge, provided a large selection of pictures showing details of Celtic fields, the strips of medieval open fields, and other features of the landscape which are now concealed below ground in some areas. Often, where sites have been inhabited for millennia, the present surface conceals the cultivation work of our ancestors. From air photographs Dr. St. Joseph was able to show the size and shape of Roman fields in the Fenlands, and of rectilinear Celtic fields underlying the ridge and furrow of Anglo-Saxon and medieval cultivation. Such features could not, of course, be seen on a modern ordnance map but are plain enough on many estate maps and surveys, enclosure awards, and so on. The air photograph can, however, illustrate these arrangements as they were, and still exist one upon another on the same piece of ground; and the pictures shown certainly brought the old systems into vivid life.

G. E. F.

THE CENTRAL VETERINARY SOCIETY

We are asked to call the attention of members to a meeting on January 8th, when the postponed scientific programme from a previous meeting will be undertaken. The meeting will be held at the Royal Veterinary College, by kind permission of the Principal, and will open at 6 p.m. (Tea at 5.30 p.m.). The main feature will be a paper by Miss O. Uvarov entitled "Corticosteroids in Veterinary Medicine." Mr. P. J. Dalton, of Salisbury, will open the discussion.

UNIVERSITY NEWS

Edinburgh

The undermentioned candidates have passed the Final Professional Examination for the degree of Bachelor of Veterinary Medicine and Surgery:—

Bewsher, Colin Berkeley; Elliott, Jean; Montgomery, John Forbes; Moody, Peter Warner; Smith, Ian Geoffrey Newlands; and Tribe, George William.

Glasgow

The following students passed in the final B.V.M.S. examination which was completed on December 9th. They will graduate on January 10th, 1959:—

Cameron, Ian D.; Hagan, Denis H.; Lopatkin, Ivan; and Pottie, Alexander D.

COMING EVENTS

January

8th (Thurs.). Additional Meeting of the Central Veterinary Society at the Royal Veterinary College, Camden Town, N.W.1, 6 p.m.

16th (Fri.). Annual General Meeting of the Mid-West Veterinary Association at the Berkeley Café, Clifton, Bristol, 2.15 p.m.

21st (Wed.) 10th Anniversary Dinner-Dance of the Centaur Society at the Berkeley, Bristol.

30th (Fri.). Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 9 p.m.

Annual General Meeting of the Yorkshire Veterinary Society in Leeds.

BRITISH VETERINARY ASSOCIATION QUARTERLY MEETINGS IN JANUARY

Tuesday, January 20th, 1959—At No. 7, Mansfield Street

5.30 p.m. Finance Sub-committee.

Wednesday, January 21st, 1959—At No. 7, Mansfield Street

10.00 a.m. Veterinary State Medicine Committee.
11.30 a.m. Home Appointments Committee.
2.00 p.m. Small Animals Committee.

Thursday, January 22nd, 1959—At No. 7, Mansfield Street

10.00 a.m. Parliamentary and Public Relations Committee.
11.30 a.m. Farm Livestock Committee.
2.15 p.m. General Purposes and Finance Committee.

Friday, January 23rd, 1959—At The Connaught Rooms, Gt. Queen Street, W.C.2

14.00 a.m. Council Meeting.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Dumfries. Hall Hill Farm, Irongray (Dec. 10).
Lancs. Marsh View Farm, Freckleton, Preston (Dec. 9).
Westmorland. Croft End Farm, Brampton, Appleby (Dec. 10).

Fowl Pest

Herts. The Old Crown, Kelshall, Royston (Dec. 9).
Norfolk. Gumshaw Hall, Needham, Harleston (Dec. 9); Green Farm, Wingfield, Diss; The Post Office, Norwich Road, Besthorpe, Attleborough; Rush Green Farm, Gislingham, Diss (Dec. 11).

Suffolk. 23, Brome Street, Brome, Eye (Dec. 11).

Swine Fever

Devon. Court Farm, Clyst St. George; Westdale Barton, West Sandford, Crediton; The Dell, Tedburn St. Mary, Exeter (Dec. 9); Aller Farm, Upottery, Honiton; Littlefields, Harepath Road, Seaton (Dec. 10); Marley Riding Stables, Marley, Exmouth (Dec. 11).

Dorset. Malpitts Farm, Shaftesbury (Dec. 9).

Essex. Fairview, Blackberry Lane, Stanway, Colchester; Allans Farm, Elmstead, Colchester; Thremhall Priory Farm, Birchanger (Dec 10).

Kent. 112, Jackson Road, Bromley (Dec. 9).

Lancs. Kesters Farm, Longton, Preston; Moss Rose Piggeries, Dickys Lane, Peel, Blackpool (Dec. 9); Inglenook Farm, Blackpool Road, Kirkham, Preston (Dec. 10); Holme Lea Farm, Whitehorse Lane, Myerscough, Preston (Dec. 11).

Norfolk. Caister Road Allotments, Great Yarmouth; The Cottage, Westhall Road, Rickinghall Inferior, Diss (Dec. 9); Priory Farm, Rickinghall Inferior, Diss (Dec. 11).

Northants. Derngate House, Welford Road, Northampton (Dec. 9).

Somerset. The Corner, Bath Road, Beckington, Bath (Dec. 9).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

English or Classical Nomenclature?

Sir,—Mr. T. Hughes Ellis's Fellowship thesis on "Obstetrics in the Ewe" is so interesting and gives us such a frank insight into his practical techniques that it invites a full discussion from all who are involved in the annual campaign of the lambing season. I should like to make some contribution to this discussion later on, but at present my attention is concentrated on the objections felt by Mr. Harvey M. F. Maynard to the use of the English word "womb" as an alternative to the Latin "uterus."

On referring to Ellenberger and Baum's, and Chauveau's, works on comparative veterinary anatomy I find that the German "die Gebärmutter" and French "la matrice" are used in their appropriate contexts equally with the term "uterus," and I believe there is generally a much greater use of native words in French and German biological literature than in English. These scientific contributions are in no way inferior in quality and it is difficult to see why a Greek or Latin designation of an anatomical feature should be held to be more scientific than an English one, always provided that the word used is precise and unambiguous.

Otto Jespersen in his "Growth and Structure of the English Language" has examined this tendency of the English to give greater prestige to words of Greek or Latin origin than to the native stock, and his observations are of great value to anyone interested in national psychology. It would appear that the reasons for this tendency are more connected with a form of snobbishness than with a concern for linguistics or scientific exactitude.

As we are talking about Welsh sheep it might be of interest to note that the commonly used Welsh word for "the womb" is "y fam" (pronounced vahm) which means precisely "the mother," a word etymologically connected with the aforementioned German and French words and with the English. I do not think that the term "uterus" has such fundamental associations.

Yours faithfully,
F. B. EDWARDS.

Newgate,
Llanbrynmair,
Mont.
December 11th, 1958.

Distemper Complex

Sir,—I was extremely interested in Mr. Bateman's letter as his figures of the success of the avianised vaccine "Epivax" confirm what I have always believed, namely, that it is as nearly 100 per cent. successful as it is possible to be.

My experience in practice has always made me disagree with people who have blamed the inefficiency of this type of vaccine for the so-called "breakdowns." I believe that in these cases the dog was incubating the disease at the time of vaccination. I have had several outbreaks in breeding and boarding kennels, and I have found that some dogs will have the disease without showing obvious symptoms, unless one makes a close examination, and these dogs are never ill in the true sense of the word.

I recently had to examine a six-month-old Boxer, and found she had badly marked teeth. There was no history of any illness, but by chance I knew that she had been reared in a kennel which had an outbreak of hard pad disease while she was there; but as far as anyone knew none of the puppies had been ill at all. Obviously this puppy had had a virus infection at an early age; it might have developed neurological symptoms subsequent to vaccination, and would then have been classified as another breakdown!

With regard to the age incidence, I have just had to put down a ten-year-old Alsatian bitch (not vaccinated) which developed severe neurological symptoms after recovery from an illness which, without doubt, was caused by the distemper-hard pad virus. This illness was contracted from the puppy next door, which had died of distemper. The bitch lived on a housing estate, so it is remarkable that she had not built up an immunity during her lifetime.

I have recently experienced quite an epidemic of the distemper complex but always in dogs which had not been vaccinated; in consequence I would be interested if Mr. Hodgman would enlarge on the remark in his letter that "all is not well."

There appears to be no dearth of distemper in areas or estates where the majority of the dogs are not vaccinated.

Yours faithfully,
H. M. PAYNE.

23, Alma Road,
Windsor,
Berkshire.
December 8th, 1958.